We claim:

1. A mutagenic oligonucleotide for site-directed mutagenesis of a double-stranded nucleic acid molecule comprising a mutagen incorporated into a single-stranded oligonucleotide having a sequence that forms a triple-stranded nucleic acid molecule with a target region of the double-stranded nucleic acid molecule.

2. The mutagenic oligonucleotide of claim 1 wherein the mutagen is selected from the group consisting of psoralen, acridine orange, an alkylating agent, a cis-platinum analog, a hematoporphyrin, a hematoporphyrin derivative, mitomycin C, a radionuclide, and a molecule that interacts with radiation to become mutagenic.

3. The mutagenic oligonucleotide of claim 1 wherein the mutagen causes a mutation in the double-stranded nucleic acid molecule in the presence of light.

4. The mutagenic oligonucleotide of claim 3 wherein the mutagenic chemical is 4'hydroxymethyl-4,5',8-trimethylpsoralen.

5. The mutagenic oligonucleotide of claim 1 wherein the oligonucleotide has a length of between 7 and 30 nucleotide bases.

6. A method for site-directed mutagenesis of a nucleic acid molecule comprising the steps of:

a) hybridizing a mutagenic oligonucleotide to a target region of a double-stranded nucleic acid molecule, wherein the mutagenic oligonucleotide comprises a mutagen incorporated into a single-stranded nucleic acid that forms a

triple-stranded nucleic acid molecule with the target region; and

b) mutating the double-stranded nucleic acid molecule.

7. The method of claim 6 comprising the additional step of activating the mutagen prior to the mutation step.

8. The method of claim 6 wherein the mutagen is selected from the group consisting of psoralen and acridine orange and is activated by light.

9. The method of claim 6 wherein the mutagen is selected from the group consisting of acridine orange, an alkylating agent, a cis-platinum analog, a hematoporphyrin, a hematoporphyrin derivative, mitomycin C, a radionuclide, and a molecule that interacts with radiation to become mutagenic.

10. The method of claim 6 wherein the mutation alters the activity of the double-stranded nucleic acid molecule.

11. The method of claim 6 wherein the double-stranded nucleic acid molecule is a gene.

12. The method of claim 6 wherein the gene is an oncogene.

13. The method of claim 6 wherein the gene is a defective gene.

14. The method of claim 6 wherein the double-stranded nucleic acid molecule is all or a portion of a viral genome.

15. A method of producing a mutagenic oligonucleotide comprising the steps of:

a) synthesizing an oligonucleotide substantially complementary to a target region of a double-stranded nucleic acid molecule; and

b) incorporating a mutagen in the oligonucleotide.

16. The method of claim 15 wherein the mutagen is covalently linked to the oligonucleotide.

17. The method of claim 15 wherein the mutagen is incorporated into the oligonucleotide during synthesis of the oligonucleotide.

18. The method of claim 15 wherein the mutagen is bound to the oligonucleotide by photoactivation.

19. The method of claim 18 wherein the mutagen is selected from the group consisting of psoralen, acridine orange, an alkylating agent, a cis-platinum analog, a hematoporphyrin, a hematoporphyrin derivative, mitomycin C, a radionuclide and a molecule that interacts with radiation to become mutagenic.